

# Stages of Decompensation in Combat-Related Posttraumatic Stress Disorder: A New Conceptual Model

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**Abstract**—This conceptual article presents a model of severe, chronic combat-related PTSD based on several years of longitudinal clinical observations of Vietnam veterans. The model describes a repeating cycle of decompensation that profoundly disrupts the veteran's life. There appear to be "stages" of decompensation that can be described clinically and may be distinct physiologically. The stages describe a wide range of functioning, from adaptive to totally dysfunctional. PTSD core symptoms, as well as several other dimensions of clinical functioning, such as affect regulation, defenses, ego states, interactions with the environment, capacity for self-destruction/suicide and capacity for attachment and insight are described for each stage. Clinical and research implications are discussed.

**Keywords**—combat-related PTSD, stages, decompensation, model

## Introduction

THE NATIONAL VIETNAM VETERANS READJUSTMENT STUDY (NVVRS) indicated that Vietnam veterans with PTSD lead profoundly disrupted lives (Kulka et al., 1990). Yet, few treatment strategies, whether behavioral or pharmacological, have shown systematic effectiveness in Vietnam veterans diagnosed with severe, chronic combat-related PTSD (Solomon, Gerrity, & Muff, 1992). Theoretical frameworks for PTSD have not led to effective clinical treatment and, consequently, successful outcomes in this population have been largely anecdotal. This article describes a conceptualization of combat-related PTSD that may stimulate innovative perspectives regarding assessment, clinical treatment, family education, treatment evaluation and research methodology. This model is based on observations of Vietnam combat veterans with severe, chronic combat-related PTSD and may or may not be relevant to other populations with PTSD.

Contributions to theories about the human response to trauma have been made by many authors, Freud (1920), Kardiner (1941), Lifton (1967), Horowitz (1978), van der Kolk, Greenberg, Boyd, & Krystal (1985) Keane, Zimering and Caddell (1985) and Green, Wilson, and Lindy (1985) among many others (for a review see Petersen, Prout and Schwarz, 1991). Contributors have emphasized specific aspects of trauma-related responses: Freud (1920) described the intrapsychic processes associated with trauma, such as overexcitation of the stimulus barrier and repetition compulsion; Kardiner (1941) used the

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### *Clinical Impressions and Longitudinal Observations*

One of the strongest clinical impressions we have had working with Vietnam veterans with PTSD is the tremendous variability in level of functioning over time. Preliminary longitudinal clinical and hormonal data from Vietnam veterans with severe combat-related PTSD suggest that both clinical and hormonal measures vary significantly during stages of highest and lowest functioning (Wang & Mason, 1995).

On one day a veteran can appear to be functioning well, is able to problem solve, to reflect on and articulate feelings, to express needs, to respond to his environment, to work toward long-term and short-term goals, to laugh and smile, to perform meaningful work, and so on. Then, two days, two weeks, two months or two years later, the very same veteran has decompensated, becoming highly reactive; unable to problem solve, unable to articulate feelings or to express needs responsibly; is rageful, irritable, paranoid, rigid, impulsive, unable to work, unable to laugh and unable to function; and is prone to abusing alcohol and other substances.

It became more clear to us that these cyclic decompensations severely disrupted the veterans' lives. We understood how these intelligent men were able to secure but also lose the twenty or thirty or forty jobs they have had, and why important relationships in their lives, with parents, siblings, friends, wives, and children, became estranged or ended painfully. We also noticed that many of the characteristics observed in Vietnam veterans with PTSD, including alexithymia, severe depression, narcissism, explosiveness, and impulsivity were significant only at certain times, suggesting that many of the labels assigned to this population might be largely state dependent.

### *Longitudinal Hormonal Observations*

After analyzing a preliminary series of longitudinal hormonal measurements from Vietnam veterans with chronic PTSD, we found that the hormonal patterns reported in PTSD, including *elevated* levels of urinary norepinephrine, epinephrine (Kosten, Mason, Giller, Ostroff & Harkness, 1987; Yehuda, Southwick, Giller, Ma & Mason, 1992), total thyroxine and total and free triiodothyronine (Mason et al., 1994) and *low* levels of urinary free cortisol (Mason, Giller, Kosten, Ostroff & Podd, 1986; Yehuda et al., 1990) were present during certain periods of observation but were less significant or absent in other periods. Fluctuations of these hormonal measurements, along with fluctuations in clinical functioning, suggested the idea of stages and also offered a possible explanation for conflicting urinary cortisol and catecholamines findings that were reported in Vietnam veterans with PTSD (Pitman & Orr, 1990).

### **Stages of Decompensation Model in PTSD**

The core idea of the model is that many Vietnam veterans with chronic, severe combat-related PTSD experience *cyclic decompensations* that are extremely destructive to all aspects of their lives, including personal relationships, the ability to work, the ability to feel, and the ability to find meaning in their lives. The recurrence of these decompensations is a major factor in the veteran's inability to function normally (e.g., keep a job and maintain relationships) and significantly obstructs rehabilitation. There appear to be stages of decompensation in which specific modes of functioning and behaviors become more prominent and in which physiologic factors may also be quite distinct. Severity of PTSD

term "physio-neurosis" to emphasize the physiological aspects of response to trauma; Lifton (1967) described the presence of a "death imprint" and survivor guilt in trauma victims; Horowitz (1978) suggested an information processing model for PTSD and proposed a five-stage process to describe the human reaction to massive stress; van der Kolk et al. (1985) developed a psychobiologic model for PTSD based on its similarity to exposure to inescapable shock; Keane, Zimering and Caddell (1985) proposed a behavioral formulation for PTSD emphasizing the role of respondent and operant conditioning in PTSD symptomatology; and Green, Wilson and Lindy (1985) developed a psychosocial model that draws attention to the interaction of several variables, such as the severity and duration of the trauma, the individual characteristics of the survivor, the role taken by the survivor and the social/cultural environments in which the trauma and the recovery take place (see Wilson, 1989, for a review).

The proposed model suggests that people with severe, chronic PTSD are vulnerable to cyclic decompensations that severely impair all aspects of functioning. At any given point in time, the individual is in a specific "stage" of decompensation. To date, the models that describe the course of PTSD culminate in some sort of resolution, usually adaptive but often pathological as well. The proposed model describes a course of PTSD in which resolution has not been reached; the individual has not been able to successfully adapt and cannot emerge from the cycle of repeated decompensations, which creates complex forms of PTSD in terms of psychological structural organization.

Horowitz's (1978) five-phase model includes outcry, denial, oscillation between numbing and intrusive states, working through, and resolution and has served as a reference for the consideration of other conceptualizations (Peterson et al., 1991). It describes an adaptive course of PTSD that ends in relative completion of the response. Epstein (1989), among others, makes the point that some people with PTSD do not follow an adaptive course, and he adds a description of maladaptive resolution of PTSD in which the person does not complete the numbing/intrusive "oscillation" phase so cannot move toward adaptive resolution of the "working through" phase. Instead, a maladaptive resolution is reached based on one of five characteristics: withdrawal, dissociation, fear, anger and embracing the trauma. Frederick (1985) also describes five phases of PTSD in combat veterans: (1) initial impact; (2) resistance/denial; (3) acceptance/repression; (4) decompensation; and (5) trauma mastery and recovery. His model, like Horowitz's, describes an adaptive process and includes a stage of decompensation as a step toward recovery. However, for many Vietnam combat veterans with severe, chronic PTSD, decompensation is not a step toward recovery, but a step toward further cyclic decompensations creating profound disruptions in their lives. Although much has been written about PTSD and the Vietnam combat veteran, relatively little work has been done describing the longitudinal decompensations or cyclic stages of PTSD in this population.

### **Background**

The model presented here was not generated on a purely theoretical basis. It is the product of observations and assessments of in-patient and out-patient Vietnam veterans at the National Center for PTSD in West Haven, CT, who suffer from chronic, severe combat-related PTSD and whose lives have been severely disrupted or have ended through accidental drug overdose or suicide. As well, it is based on the evaluation and treatment of hundreds of Vietnam combat veterans seen in private practice at the Stress and Trauma Center from 1973 to the present in Cleveland, Ohio.

**TABLE 1**  
**Stages of Decompensation in Combat-related PTSD**

	CLINICAL SYNDROME			ADAPTIVE FUNCTIONING		
	PTSD Symptoms	Affect Regulation	Defenses	Ego States	Facial and Body Movements	Interactions with the Environment
STAGE 1: ADAPTIVE GOOD TO MAXIMUM FUNCTIONING Clinically Stable GAF Estimate 55-75	Minimal to moderate adequate sleep; minimal numbing; limited hyperarousal; maximal memory and concentration.	adequate, responsible expression of affect; maximum range of affect; limited anxiety; limited paranoia	most adaptive; may include sublimation, intellectualization, rationalization, repression, altruism	adequate self esteem; ego integrity; intact ego boundaries; clear sense of self in the future (hope); maximum self- other distinction; maximum self- sense of control and belonging.	spontaneous facial expressions; fluid body movements; smiles; laughter	flexible; able to give and receive information; genuine humor; avoids pain; able to problem solve; can be creative; responds to positive reinforcement.
	moderate to severe; intrusions; nightmares; startle responses; hypervigilance; mood swings; irritability; detachment; memory and concentration ↓ sleep interest in activities	inadequate; must work at containing; restricted range of affect; anxiety; paranoia; twittrawaw impulsiveness thomicidity ↑ desire for drugs and alcohol;	less adaptive; projection; splitting; denial; disavowal; aggression; numbing; fixation; alexithymia; anhedonia; and narcissistic entitlement	inadequate self esteem; vulnerable ego boundaries; deteriorating ego boundaries; vague sense of self in the future (losing hope); blurred self-other distinction; sense of losing control; sense of alienation (origin threat)	decreased range of facial expressions; rigid body movements; smiles (physical complaints, e.g. headaches)	more guarded and rigid; less able to give and receive information accurately; decreased responsibility yet increased reactivity; withdrawal; isolation; narrow focus; degrading humor; responds to negative reinforcement
STAGE 3: DECOMPENSATION DYSREGULATED DISCHARGE STATE	Flashbacks; ↑ startle ↑ dissociation; ↑ anger	absent; unmanaged affect; overwhelming anxiety;	inadequate; primitive; maladaptive; destructive; impulsive acting out; regression; ↑ dissociation	no self esteem; fragmentation of self; disintegration of ego boundaries; no sense of self in future (no hope); absence of self-other distinction; identity confusion; dissociation; total loss of control;	variable facial and body movements	signs of loss of contact with the environment; extremely narrow focus due to recklessness; no self care
VERY POOR FUNCTIONING Clinically unstable GAF Estimate 0-35		SENSE OF rage; violence; blackouts; paranoia; extreme impulsivity; craves adrenaline rush; enjoys the pain; the feeling of battle; binge drinking and drugging; labour is exciting		OR		ISOLATION total "shut down"; total numbness; unresponsiveness to others; isolation; dissociation; stays in "bunker"; cannot engage in interpersonal interactions
STAGE 4: DEPRESSION/ HOPELESSNESS DYSREGULATED AFFECTIVE STATE	variable symptoms; ↑ startle/hyperarousal	inadequate; profound sadness; hopelessness; quiet; humiliation; anger at self; emotional pain	inadequate; maladaptive; self hated	very poor self esteem; vulnerable ego boundaries; fragile ego boundaries; very limited sense of self in future (little or no hope); weak self-other distinction; very little sense of control; sense of alienation (origin shame)	decreased range of facial expressions; slowed body movements	limited ability to give and receive information; broader focus; isolation; ↑ reactivity and reactivity
Clinically unstable POOR FUNCTIONING GAF Estimate 0-45		REGROUPING looking for reasons to live; willingness to try again and start over; humility; accepting limitations and disabilities; accepting support; transition to stage 2		OR		GIVING UP no future; no answers; no hope; tired of the neverending destructive pattern; shame; unworthiness; feelings of utter failure; contempt for self and for society; desire to rid self and others of pain by committing suicide; despair; returns to stage 3 or attempts suicide

symptoms are related to the stages of decompensation, but several other factors, such as affect regulation, interactions with the environment, defenses and capacity for attachment and insight also define the stages. The stages are described in Table 1 and are summarized as follows:

*Stage 1: Most Adaptive (good to maximum functioning)*

In this stage, the PTSD patient can appear to be very normal. His interactions with the environment are somewhat flexible. He can interact with others appropriately, give and receive information, express affect responsibly, problem solve, work toward goals, laugh, be creative and respond to positive reinforcement. In Stage 1, several positive qualities are at a maximum: ability to reflect on feelings and behavior, responsiveness, breadth of focus, concentration, memory, interest in activities, range of affect and affect regulation, importance of relatedness, consideration of others, fluidity of body movements, and spontaneity of facial expressions. Most symptoms, if present, are at a minimum: intrusions, avoidance and numbing, hyperarousal, sleep disturbance, anxiety, paranoia, suicidality, dissociation and desire for drugs or alcohol. This is the stage in which the patient is able to get a job or attract a spouse. He is able to engage in treatment and at times can feel to the clinician like more of a peer than a patient. However, it does not last. Soon, triggered from an internal or external source (anniversaries, an interpersonal conflict, news events, a particularly bad nightmare, etc.), the patient feels threatened, anxiety increases and he starts to enter the next stage and to begin a cycle of decompensation.

*Stage 2: Survival (fair to poor functioning)*

In this stage, the orientation is primarily defensive. The patient begins to constrict, physically and emotionally. The breadth of focus of Stage 1 narrows considerably. Intrusions, avoidance and numbing and hyperarousal increase, especially sleep disturbance. Interactions with the environment are more guarded and cautious; there is less ability to give and receive information as the preoccupation with survival is paramount. Body movements become more rigid; facial expressions lose spontaneity; range of affect is restricted; humor, if at all present, becomes degrading of others; paranoia increases as well as mistrust of authority figures (and the VA); the importance of relatedness and consideration of others decreases and the ability to reflect on behavior and feelings disappears. Interest in activities, memory and concentration decrease. Affect regulation is inadequate; reactivity is high; irritability and anger grow; containment is hard work and the urge to isolate becomes strong. The patient is more sensitive to negative reinforcement; i.e., responses that may temporarily alleviate the aversive internal state are strong, such as impulsivity toward risk taking and the desire for drugs or alcohol. Suicidal and homicidal ideation increase. The patient has lost some contact with the environment and may experience mild dissociation at times. All of these symptoms are exacerbated by lack of sleep. (See Table 1 for a capsule summary.)

*Stage 1/Stage 2 Equilibrium and the Stage 2 Threshold Point (Figure 1)*

Most veterans with chronic, severe combat-related PTSD are in Stage 2 most of the time. These men can vacillate between early Stage 2 and Stage 1 if they are in a safe environment, get adequate sleep, experience emotional support and stay sober. In fact, a

*Stage 3: Decompensation (very poor functioning)**Sensation seeking or Shutting down: Dysregulated Discharge State*

In this stage, meaningful contact with reality is lost. Anxiety is overwhelming, and affect is totally unmanaged. Containment is no longer possible. At this point, the veteran's focus is extremely narrow. There is no concept of a future, so responsibilities and consequences exert little influence. The veteran feels that there is nothing to lose, so his behavior can become exceedingly reckless. The risk of death as a result of reckless behavior is highest in this stage (Wilson, 1988; Wilson, Harel & Kahana, 1988). Two typical ways that veterans decompensate are: 1) *sensation seeking*: high risk, violent, angry, or impulsive behavior, excessive spending, craving for an adrenalin rush, binge drinking and/or drug-ging, (veterans call this a "run"); or 2) *totally shutting down*: going into their "bunker" or the woods or disappearing for an extended period of time, unable to interact in a meaningful way with anyone. Often in this stage flashbacks, hallucinations, blackouts and other dissociative experiences are most intense. Relatedness and consideration of others are irrelevant, often leading to behavior that cuts off most or all of the existing support the veteran had managed to build. The thread that had previously connected him to anything meaningful in his life (relationships, job, financial security, self-respect) has been severed and is at risk to be damaged or destroyed permanently.

Support and a feeling of connectedness and familiarity are powerful forces preventing decompensation. The tragic part is that veterans with chronic PTSD often destroy or unconsciously sabotage the support and connectedness they desperately need when they start to decompensate. They close the door on the very resources that could help them regain stability. This form of pathological detachment is often an attempt to gain control over dysregulated states by isolation and by being on a solo search and destroy mission. Clearly, the consequences are precisely the opposite and are profoundly maladaptive.

*Stage 4: Depression/Hopelessness (poor to very poor functioning)**Regrouping or Giving up: Dysregulated Affective State*

When the storm is over, and the veteran has ended his "run," he is tired. His focus broadens again, but now all he sees is devastation and failure. He feels sick, feels revulsion over what has happened. He thinks "My God, what have I done . . . the people I've hurt, the job I've lost, the trust I have betrayed, the money I've spent, the taboos I have broken, the lives I have endangered." He starts to obsess over his failings, hating himself, feeling helpless, feeling hopeless, humiliated, defeated or simply confused. Reactivity and startle are at their lowest point. A deep depression ensues, with psychomotor retardation, guilt, emotional pain, depressed appetite, longer periods of but still irregular sleep, no interest in activities, and increased difficulties with concentration and memory. There is a strong desire to isolate but, in contrast to stage 2, this isolation is not the result of not caring about others or wanting to be distant. On the contrary, the veteran is very vulnerable and needy at this point but feels totally unworthy of anyone's support or love. The highest risk of premeditated suicide occurs in this stage. The suicidality is intensified, not because PTSD symptoms are intolerable, but because the veteran feels absolutely useless, defeated, unworthy of love or respect and unable to function as a man, a husband, or a father as a result of the repeated decompensations he has experienced. At this point, the veteran typically chooses one of two paths: a) *regrouping*: if he has access to treatment or other support, he can start to regroup, (as in Vietnam) looking for reasons to live, accepting limitations and

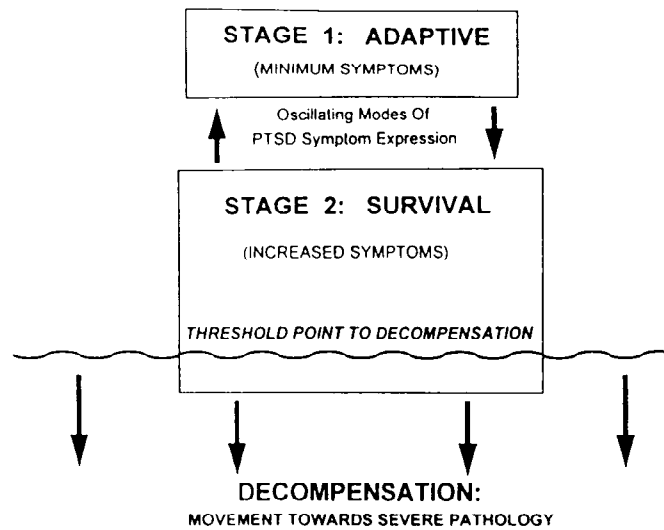


FIG. 1. The Stage 1/Stage 2 equilibrium and the Stage 2 threshold point to decompensation

goal of rehabilitation could be seen as maintaining the equilibrium between Stage 1 and early Stage 2.

However, if a certain threshold point in Stage 2 is reached, the transition back to Stage 1 becomes nearly impossible, and the veteran can quickly move toward decompensation (Stage 3). For successful reversal back to Stage 1, two critical factors seem to be important to address: sleep and support. Adequate sleep is consistently problematic for chronic PTSD patients, and at the threshold point in Stage 2, sleep deprivation is typically even more pronounced. The lack of sleep accelerates the progression toward decompensation and, if not treated, will greatly decrease the chances of a successful reversal to Stage 1.

Psychological support and staying connected to people is another critical factor in turning the tide away from decompensation and toward stability. Accepting support by the veteran is difficult at this time because he has a strong desire to isolate and detach from those around him. Wilson (1980) characterized this stage as *survivor mode functioning*, strongly reminiscent of behavioral coping in the jungles of Vietnam. As a form of PTSD this is a regression and type of behavioral reenactment. He is less considerate of others and places less importance on relationships (because survival is his focus), so people around him are less likely to offer support. The veteran needs a safe place, not too much stimulation and support that does not demand responsiveness from him, (e.g. a card, a short, positive conversation, listening, quiet company while watching TV, etc.). It is a delicate time because he is teetering on a thin line between decompensation and regaining stability. Too much interaction will force him to withdraw more, feeding his anger, impulsivity, numbness and "shutting down," leading to decompensation. Too little interaction will allow the pattern toward decompensation to continue without obstruction.

When the threshold point in Stage 2 is reached and not successfully managed, the forces toward decompensation are tremendous. One veteran describing the pull toward sensation seeking during decompensation said, "The force is so strong, I feel like a moth being attracted to a flame," and yet another said "It's like being in Nam again . . . wired, tired and ready. It don't mean nothin' man."

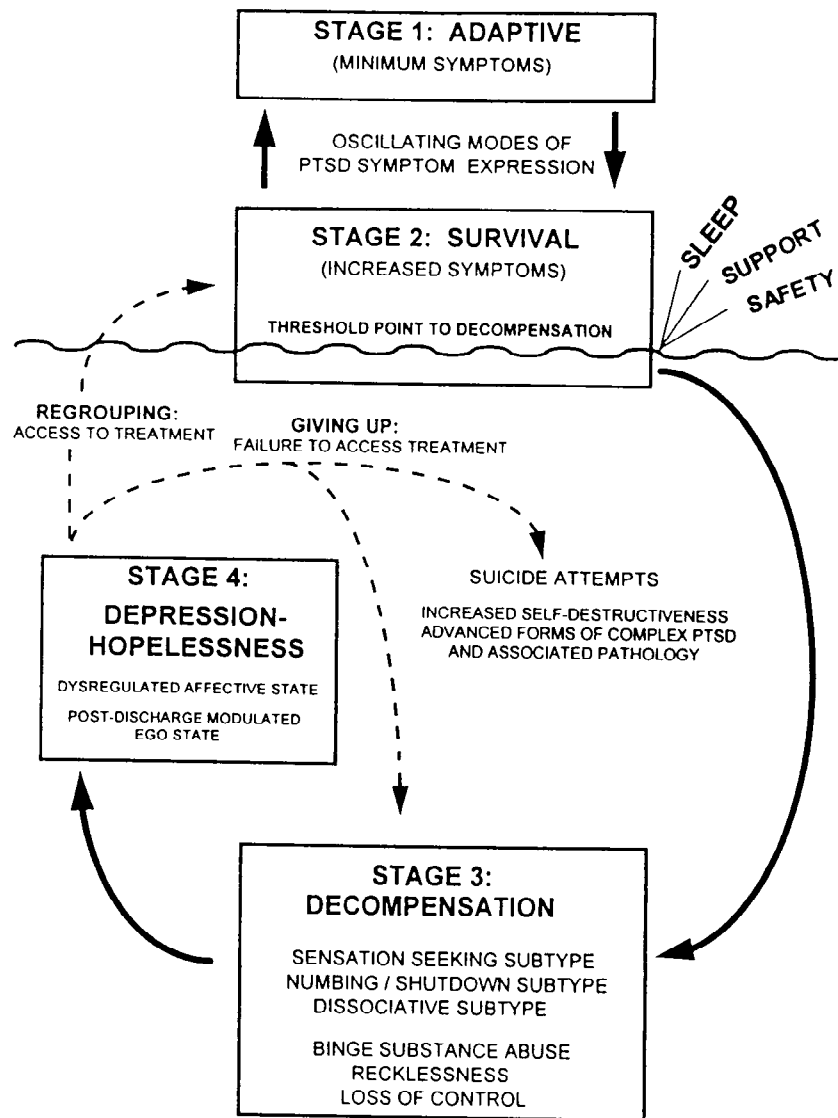


FIG. 2. Progressive stages of decompensation and recompensation in severe combat-related PTSD

The stages of decompensation represent a general model based on longitudinal observations of veterans over several years. Of course, there are many variations to the cycle. Some veterans may not go through the stages in the same order or may skip a stage altogether. However, the stages are presented in a way that dynamically describes the decompensation cycle of many of the veterans observed.

### Clinical and Research Implications of the Stages of Decompensation Model

If the stages of decompensation model is a useful and accurate way to describe the longitudinal course of severe, chronic, combat-related PTSD in Vietnam veterans, the



accepting support, and he will likely transition to Stage 2; or b) *giving up*: if he does not access treatment and remains in this hopeless, despairing state, he will either become reckless again and go back to Stage 3, or attempt suicide, directly or indirectly (see Wilson and Zigelbaum, 1986, for a discussion of the depression-suicide sub-type of survivor mode functioning). All four stages of the decompensation cycle are diagrammed in Figure 2.

### Longitudinal Features of the Cycle

This cycle of decompensation repeats itself unless the veteran can learn to expect and manage the changes in mood and behavior that will inevitably occur. Clinicians may be unaware of the power of the cycle and may expect the veteran who is feeling well (Stage 1) to be able to maintain his adaptive, functional behavior. However, the nature of PTSD is that events, anniversaries, and other external or internal stimuli will result in increased intrusions, arousal and avoidance, and the veteran will inevitably enter Stage 2. At this point, the variables that controlled his behavior in Stage 1 may no longer be effective. A whole new set of variables, events and feelings now control his behavior. Veterans have reported that, as they enter the constriction of Stage 2, many things change: what they want to wear, what they want to watch on TV, what activities they find compelling, and so on. In Stage 2, the veteran responds more strongly to negative reinforcement, so that anything (drugs, alcohol, gambling, other impulsive behavior) that relieves his aversive internal condition is more powerfully sought.

The change from Stage 1 to Stage 2 represents not only an increase in PTSD symptoms but also a change in the whole reinforcement system affecting the veteran. In treatment, it may be useful to identify the different reinforcement systems and to shift the focus from PTSD symptoms alone to a more organismic view of the PTSD patient, attending to changes in functional capacity, regulation of affect, interactions with the environment, risk of self-destructive behavior, and strength of particular behavioral repertoires.

Wilson and Zigelbaum (1986) describe their observations of altered states or modes in individuals with PTSD as follows:

In our clinical and forensic work with survivors of profoundly stressful life events, we have observed that because of ideational or environmental stimulus, the individual with PTSD may enter into a *survivor mode* (Figley, 1978; Wilson & Zigelbaum, 1983) of functioning, which is characterized by some or all of the following qualities: an altered state of consciousness, hyperalertness, hypervigilance, excessive autonomic nervous system arousal, frenetic behavior, paranoid ideation, mistrust, and the use of survivor skills and cognitive capacities learned during the period of the traumatic episode. *In addition, depending on the particular personality characteristics of the survivor-victim and the situational stressors that trigger the onset of the survivor mode, the person may experience an oscillation between the survivor mode of functioning and normal personality functioning. Typically, this occurs in a dissociative reaction, but it can occur in nondissociative survivor modalities.*

It is critical for clinicians to plan a strategy with the veteran for all aspects of the cycle, including the survival stage, decompensation and the following depressive/regrouping stage. If there is no plan allowing for decompensation and recompensation, the veteran's feelings of failure and inadequacy are even greater because now in addition to his family, employer, and friends, he has disappointed his therapist.

### *Medications*

If Vietnam combat veterans with chronic, severe PTSD do experience stages of decompensation that have distinct clinical and physiological markers, then the *lack* of efficacy of medications across stages may be understandable and predictable. It is not uncommon for veterans with chronic PTSD to have tried many medications of several different classes without consistent symptom relief over time. A typical complaint among veterans and a common observation of clinicians is that a particular medication works for a while, but then it becomes ineffective or sometimes makes symptoms worse. It is possible that a certain medication works well only in a particular neurophysiologic state and stage of PTSD. When a change of stage occurs, the medication may not be effective in the neurophysiologic state that correlates with the new stage. In this regard, PTSD must be considered as a dynamic process rather than as a static entity. The configuration that constitutes the internal structure of PTSD changes in an interactive manner between situational, trait and state dependent variables. It may be that, in order to treat PTSD more effectively pharmacologically, stages in PTSD need to be evaluated and then medications adjusted accordingly.

The notion of stage specific medication has some support from a small sample of in-patient veterans at West Haven VAMC who presented with increased dissociation and impulse dyscontrol (consistent with Stage 2, near the threshold point as described in the model) and who rapidly recompensated when treated briefly with a low dose neuroleptic (Rasmusson, Bergherr & Lubin, personal communication). Treating the exacerbated sleep disturbance prior to decompensation is another example of time limited, stage specific pharmacological intervention that may prevent decompensation.

### *Insight*

The motivation among many veterans with chronic, severe PTSD to understand their disorder and figure out how to manage it is remarkably high. The ability to insightfully describe their feelings, thoughts and behavior can be well developed in these veterans; however, reflection is maximal only in Stage 1 or very early Stage 2. At any other point in the cycle, they cannot reflect as well because they are overwhelmed with either defensive operations, dysregulated discharge or dysregulated affect. Therefore, it is important to discuss and work out a plan for management of all aspects of the decompensation cycle while the veteran is in the equilibrium between Stage 1 and early Stage 2.

### *Cognitive Changes*

Difficulty concentrating is one of the PTSD symptoms listed in the hyperarousal cluster of the DSM IV and is a common complaint among veterans with PTSD. Our clinical observations also support frequent reports of problems with memory, aside from memory regarding the trauma, in this population. Bremner et al. (1993) reported that Vietnam veterans with PTSD scored lower on neuropsychological tests of memory than did a comparison group. Yehuda et al. (1995) did not find global memory deficits but found specific deficits in monitoring and regulation of memory information in combat veterans with PTSD compared to a matched control group. The stages model would suggest that cognitive functioning, including memory, concentration and problem-solving fluctuates in different stages and the fluctuations should be evident in neuropsychological assessments

implications of the model are both significant and far reaching. Some examples of issues or concepts for which the stage theory may have relevance or parctical implication are now presented.

#### *Posttraumatic Decline*

Posttraumatic decline as a result of massive psychic trauma has been described by Titchener (1986):

Massive psychic trauma are capable of devastating even the most secure and mature. These experiences, particularly when they are not worked through after the initial shock, often eventuate in a chronic syndrome we term *posttraumatic decline*, which results in the removal of the person from meaningful participation in family, society, work and all forms of gratification.

Repeated decompensations certainly contribute to low social, interpersonal and occupational functioning and may be the force driving posttraumatic decline. The stage theory would suggest that, in veterans with chronic combat-related PTSD, posttraumatic decline is related to the cumulative effect of a series of decompensations and that a major focus of treatment should be shifted to the management of this destructive cycle of decompensation.

#### *State Dependent Learning*

Wilson and Walker (1986) propose that state dependent learning theory may be relevant to PTSD. According to the theory, information, behavior and coping skills encoded and/or reinforced in a state of heightened autonomic nervous system arousal or other specific physiological states will be retrieved when those states are once again induced. This idea is at the core of the stages model. Different stages manifest specific behaviors, cognitive and emotional processes, defenses, experience of self and interpersonal behaviors. For example, it has been reported that veterans with PTSD use more emotion-focused coping than do veterans without PTSD (Blake, Cook & Keane, 1991; Fairbank, Hansen & Fitterling, 1991). The model suggests that emotion-focused coping is prominent in Stages 2, 3 and 4, but if the same veterans were assessed in Stage 1, emotion-focused coping would be less prominent and problem-focused coping would be at a maximum.

Similarly, Keane et al. (1985) proposed a behavioral model for PTSD suggesting that a conditioned emotional response to stimuli that have been previously paired with aversive stimuli may account for many of the symptoms in PTSD. In the stages model, the behavioral formulation would be most relevant in Stage 2 and less relevant in other stages. Alexithymia and narcissism, considered to be trait characteristics, have been observed in PTSD patients (Krystal, 1982; Hyer et al., 1990). The stages model suggests that these qualities may be state related. Our personal observations of veterans with PTSD over time support the notion that the inability to recognize and express emotion (alexithymia) and narcissism are definitely more intense in Stages 2 and 3 and decrease (sometimes dramatically) in Stage 1. What this suggests is that specific affective-cognitive states underlie a broad range of personality processes influenced by post-traumatic conditions.

& Hammett, 1985; Southwick, Yehuda & Giller, 1991). According to the stages model, it is highly likely that depression would be diagnosed if assessed in Stage 2 or Stage 4. Since we are proposing that depression is part of the cycle in PTSD, not a separate disorder, its presence should not qualify it as a comorbid diagnosis according to this model. This is not to say that the affective disorder component in PTSD should not be assessed or identified. On the contrary, the affective disorder component in the veteran with PTSD is an important aspect to evaluate. Our observations suggest that there may be a subset of patients who have a stronger affective disorder component in their cycle that should be identified and considered when evaluating suicide risks and when choosing medications or other treatments. As noted by Wilson (1989), the underlying issue of co-morbidity concerns the unique neurological pathway of PTSD and how it differs from true affective disorders of endogenous origin.

#### *Treatment Evaluation*

Typically, evaluation of treatments for PTSD consists of measuring PTSD symptoms at point "a" before treatment and at point "b" after treatment. If the stage theory is accurate, then it is likely that measuring only PTSD symptoms will not give the best information regarding the effects of treatment. Instead, evaluating *modifications of the cycle*, for example, duration and intensity of stages of decompensation or periods of maximum functioning within the cycle, may give a better indication of overall progress. A patient who has made considerable progress in preventing decompensations still has many days in which symptom levels are fairly severe. An assessment of symptom severity would not necessarily reflect the improvement of quality of life and overall functioning he has achieved. In addition, the lack of systematic evidence for effective treatments may be in part due to the fact that research subjects in treatment studies are in a variety of stages at baseline. The model suggests that the same veteran will probably respond differently to a particular intervention in Stage 1, 2, 3, or 4. Clearly, these are empirically testable phenomena that could advance the methodology of treatment outcome studies.

#### *Service Connection/Compensation Evaluation*

The stages model helps to explain why the area of service connection and compensation hearings has been so conflictual. Severely disabled veterans who are not able to work can appear normal and engaging (Stage 1) in a compensation interview. Their work history and longitudinal functioning are poor but in the interview their disability is hidden. Often, a veteran who happens to be functioning well on the day of a compensation hearing and who is denied benefits becomes rageful and says "What do I need to do, go in there and threaten to kill the guy to let him know how sick I am?" The recognition that chronic severe PTSD is a disorder that makes one vulnerable to cyclic decompensations and unable to function normally may help examiners and others understand that it is possible to appear normal at a specific point in time and yet not be able to function normally.

#### *Simple and Complex PTSD*

Not everyone with PTSD decompensates repeatedly. Most people who develop PTSD from circumscribed traumatic events, and who have adequate support, vacillate between Stage 1 and Stage 2; they experience symptoms, but they do not become chronically

administered in different stages. Our observations support the notion of maximum cognitive functioning in Stage 1 and decreased cognitive functioning in Stages 2–4. Often, as veterans move toward decompensation, they are more likely to lose keys, forget appointments, have difficulty reading, watching a whole television program or comprehending what was said in a class or discussion or treatment. In our model, neurocognitive impairment is a manifestation of hormonal, autonomic, and psychological processes that affect specific intellectual capacities.

### *Clinical Intervention*

Given the extensive changes in affect regulation, cognitive functioning, reinforcement systems and capacity for insight across stages, it follows that the most effective clinical interventions might include an assessment of stage and an appropriate adjustment to accommodate that stage. Many sensitive clinicians are already functioning in this way, and the model could help to explicitly describe stages, making the process more conscious and consistent. Stage 1 may call for actively encouraging the patient to examine and reflect on his behavior in different stages and to create strategies for the next difficult period as well as to plan for continued good periods, maximizing the use of problem focused coping and problem-solving skills. The veteran in this stage is most adaptable so acquiring and maintaining methods to decrease arousal, such as relaxation, group support, structured activities, safe interpersonal interactions and good sleep habits are very important. Direct confrontation about past behavior can be effective because relatedness and future events can influence his behavior in this stage.

Stage 2 brings with it less capacity for insight, decreased affect regulation, increased intrusions, avoidance, hyperarousal, impulsivity and urge for drugs and alcohol, so interventions may be more successful if they are more immediate, concrete and specifically related to coping with the present aversive internal state. Different strategies may be effective for different veterans, including respite, temporary pharmacological intervention, and using the methods developed and practiced in Stage 1 to decrease arousal. As mentioned above, two key factors that appear to be important in preventing decompensation are staying connected to others and getting adequate sleep.

The internal stimuli experienced by the veteran at the threshold point of decompensation in Stage 2 has typically been followed by dysregulated emotional discharge, excitement, destruction, despair and loss (Stage 3). It will take several trials of successful prevention of decompensation to establish a sequence that includes dysregulated affect, increased symptoms (Stage 2) and then *recompensation* (Stage 1). The prevention or modification of the duration and degree of decompensation has a direct effect on the intensity of the defeat, desperation, depression and suicidality experienced in Stage 4. The capacity for premeditated self-destructive behavior increases dramatically in the aftermath of a decompensation. Therefore, a major portion of treatment of chronic, severe PTSD should not focus on alleviation of PTSD symptoms alone, but should also include work on the other, more modifiable, factors that contribute to the devastating cyclic decompensations that destroy the integrity of the individual.

### *Comorbid Depression*

High rates of comorbid depression in PTSD patients have been reported in several studies (Sierles, Chen, McFarland & Taylor, 1983; Davidson, Schwartz, Storck, Krishnan

sive, and threatening. The stages model could be helpful in educating families to recognize the changes in mood and behavior that will occur in the veteran. It can also provide a framework family members can use when creating strategies to cope with these changes. Strategies would include family members identifying the veteran's behaviors associated with certain stages, finding out how best to respond (or not respond) to the veteran in those stages and, most importantly, how each family member can protect him/herself and address his/her own needs as the veteran passes through these stages.

### Future Research

We are in the process of exploring hormonal correlates associated with the stages of decompensation in PTSD, with the hope of identifying the biological patterns related to clinical symptoms. This link would greatly enhance our understanding of the dynamics of the disorder and may also provide a rational basis for the choice of medications or other treatments in different stages.

Since treatment strategies for chronic combat-related PTSD have not proved to be systematically effective, more intensive longitudinal data in general, whether clinical or biological, are extremely important to collect in order to provide clues about how, why and to what extent symptoms and overall functioning change in these patients, as well as what interventions at what particular points in the cycle appear to be effective. It is clear that we need new perspectives in approaching PTSD, and this model attempts to broaden the current focus, both in terms of suggesting increased longitudinal observations of PTSD patients and in terms of examining *important clinical dimensions beyond PTSD symptoms alone*. We strongly suggest the collection of longitudinal data on PTSD patients at several points in time during periods of maximum and minimum functioning to add to our knowledge of the dynamic aspects of the disorder and to provide data to more thoroughly evaluate the proposed stages model. We invite those who have experience and expertise in treating other populations with PTSD to determine the extent to which this model may be useful with different trauma survivor groups.

### Summary

Many Vietnam veterans with severe chronic PTSD still lead profoundly disrupted lives. Cyclic decompensations significantly impair the veterans' occupational, social and interpersonal functioning; yet, between decompensations, there can be periods of fairly good functioning. Current treatment strategies do not show systematic effectiveness in treating severe, chronic combat-related PTSD, indicating the need for new approaches to understanding the disorder.

The proposed model includes stages of decompensation that describe the extremely variable levels of functioning observed in these patients, and that also provide a context not only for fluctuating severity of PTSD symptoms but also for variability in *several other important clinical dimensions* including *affect regulation, interactions with the environment, coping styles, cognitive functioning, defenses, ego states, suicidality and capacity for attachment and insight*.

Preliminary longitudinal hormonal studies suggest that biological fluctuations also occur across stages that may prove to be an important adjunct in understanding and characterizing the stages in the cycle of decompensation.

This model introduces "stages of decompensation" as a new conceptualization of

dysfunctional. In time, many of these people come to an adaptive resolution, as described by Horowitz's model. Herman (1992) refers to this type of PTSD as "simple" PTSD. Perhaps, whether one stays in the equilibrium between Stages 1 and 2 or experiences the whole cycle including decompensations is one of the differences between "simple" and "complex" PTSD as described by Herman (1992):

The syndrome that follows upon prolonged, repeated trauma needs its own name. I propose to call it "complex post-traumatic stress disorder." The responses to trauma are best understood as a spectrum of conditions rather than a single disorder. They range from a brief stress reaction that gets better by itself and never qualifies for a diagnosis, to classic or simple post-traumatic stress disorder, to the complex syndrome of prolonged, repeated trauma.

Herman's clinical description of complex PTSD includes alterations in affect regulation, alterations in consciousness, alterations in self-perception, alteration in perception of perpetrator, alterations in relation with others and alterations in systems of meaning. All of these characteristics are present in the more dysfunctional stages described by the model. More recently, Wilson (in press) has suggested that PTSD should be viewed as a distinct psychiatric syndrome in which the core injury is to the individual's ego states that results in a re-configuration of personality processes and psychobiological adaptation.

### *Rehabilitation*

According to the model, a goal of rehabilitation in chronic severe PTSD would be to manage the decompensation cycle effectively so that complex PTSD, so to speak, can become more like simple PTSD. That is, to find a strategy that keeps the veteran functioning in the equilibrium between Stage 1 and Stage 2. The veteran, with the help of clinicians, psychiatrists, family members, etc., must learn to develop strategies that allow him to tolerate symptomatic cognitive/emotional/physiological states without decompensating. This strategy may involve adjusting medications, respite time, structured activity, therapeutic interactions, development of trust in at least one relationship, relaxation techniques, exercise, group support, and minimizing the use of maladaptive emotion focused coping while maximizing the use of problem focused coping, even when affect regulation is inadequate. Symptomatic episodes need to be reframed as signals to invoke successful arousal reducing techniques instead of being interpreted as signs of unavoidable decompensation and destruction. Stated differently, recognition of the stages and cycles of decompensation processes may allow for targeted clinical interventions that are efficacious and healing in nature.

### *Family Education*

Impairment of interpersonal functioning that causes difficulty in maintaining meaningful relationships is a major problem affecting the quality of life for many Vietnam veterans with PTSD. According to the NVVRA, 70% have been divorced, and the majority of Vietnam veterans with PTSD have significant relationship problems (Kulka et al., 1990). The difficulties of living with a person who passes through stages of decompensation can be paramount. At one moment, the veteran is responsive, considerate, and able to think clearly, and then suddenly he becomes enraged, aggressive, irrational, insensitive, impul-

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chronic, severe combat-related PTSD that could lead the way to innovative perspectives regarding assessment, pharmacological and clinical interventions, family education, and research strategies.

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